Long term health status of children recovering from severe acute malnutrition

Briend, André; Berkley, James A

Published in:
The Lancet Global Health

DOI:
10.1016/S2214-109X(16)30152-8

Publication date:
2016

Document version
Publisher's PDF, also known as Version of record

Long-term health status of children recovering from severe acute malnutrition

Published Online
July 25, 2016
http://dx.doi.org/10.1016/S2214-109X(16)30152-8

See Articles page e654

Severe acute malnutrition (SAM) is a life-threatening condition that often occurs during a critical period for a child's growth and development. Treatment of SAM is among the most cost-effective interventions to prevent childhood death.[1] Thanks to the rapid expansion of community-based treatment programmes worldwide, every year millions of children are treated for SAM.

The Article by Natasha Lelijveld and colleagues in *The Lancet Global Health*[2] shows that, despite treatment, the long-term survival and health of children who were previously treated in hospital for complicated SAM is suboptimum, and that more attention needs to be given to risks of chronic diseases later in life. As pointed out by the authors, this observational study does not establish a cause–effect relationship since chronic problems might be related to conditions that existed before the episode of SAM. For example, children in the case group were already severely stunted at the beginning of treatment for SAM without recovery during treatment,[3] thus their shorter height-for-age later in life cannot be fully ascribed to the episode of SAM. Another childhood condition with long-term consequences is low birthweight, which has long-term consequences for chronic diseases and is a risk factor for malnutrition.[4] Long-term effects are likely to be mediated by epigenetic changes arising from exposures that might be transgenerational or occur preconception, in utero, or in early life.

The finding that children treated for SAM had a reduced proportion of lean tissue, which might predispose them to chronic diseases later in life, cannot be ignored. The study's results suggest that abnormal body composition is the result of a deficit in lean tissue rather than of excess fat tissue deposition. Lelijveld and colleagues' findings are consistent with those of previous studies that examined children with SAM at the end of their treatment period, which also reported suboptimum recovery of lean tissues.[5] This effect is unlikely to be related to the high fat content of diets used for nutritional rehabilitation such as the currently used ready-to-use therapeutic foods (RUTF), which provide more than 55% of their energy via fat. This proportion is similar to that of breast milk and reflects the high energy requirements of rapidly growing children who need to accumulate fat, which is the case for both children recovering from malnutrition and for healthy infants during the first months of life.[6] The deficit in lean tissue is more likely to be related to an insufficient intake of the nutrients needed for lean tissue growth either during the treatment of the episode of SAM itself, or more importantly, during the following months when the child returns to relying on a family diet with limited dietary diversity and low intake of foods from animal sources.

Lelijveld and colleagues suggest that some catch-up growth occurs after discharge, even outside the expected 1000-day window, in the absence of any specific intervention. The real question now is to establish how this spontaneous partial recovery after treatment for SAM can be improved. During treatment and convalescence, the proportion of dietary intake consisting of nutrients needed for lean tissue growth might also affect body composition. Among the possible factors that could limit lean tissue synthesis, the role of high-quality dietary protein might have been previously underestimated, as suggested by the results of another study,[7] which showed an association between circulating essential amino acids and linear growth. The availability of dietary proteins might be particularly limited for these children. Their requirements might be greater than they are currently estimated to be, resulting from the presence of impaired gastrointestinal function and chronic inflammation. Insufficient zinc intake is also associated with reduced lean tissue deposition and height growth.[8] The possibility to improve the long-term recovery of lean tissue by increasing the intake of high quality proteins, zinc, or both, as well as other micronutrient intake should be explored.

The poorer school performance of children treated for SAM compared with the control groups is also a serious concern, and might also be related to chronic stunting, poor quality diet, or unfavourable social environments before and after the SAM episode. Possible approaches to improve current management should be explored. Results from two clinical trials have suggested that the essential fatty acid composition of RUTF should be reconsidered, which has implications for cognitive

development.[9] Additionally, evidence from previous small-scale studies has suggested that psychosocial stimulation is important for cognitive development during recovery and follow-up.[10] This finding should be confirmed in larger efficacy studies and ways to improve the integration of this component into existing large-scale programmes should be explored during treatment and follow-up.

The important concerns raised by Lelijveld and colleagues should be balanced against the lives saved by SAM programmes. These programmes should be expanded given their overwhelming short-term benefits. However, these findings should prompt new research to improve the management of SAM. The conclusion of this paper, that it is vital that SAM-survivors should thrive, not just survive, should be fully endorsed.

André Briend, James A Berkley
Tampere Centre for Child Health Research and Tampere University Hospital, University of Tampere, Tampere, Finland (AB); Department of Nutrition, Exercise and Sports, Faculty of Science, University of Copenhagen, Frederiksberg DK-1958, Denmark (AB); Centre for Tropical Medicine & Global Health, Old Road Campus, Oxford, UK (JB); and KEMRI/Wellcome Trust Research Programme, Kilifi, Kenya (JB)
andre.briend@gmail.com

We declare no competing interests.

1 Bhutta ZA, Das JK, Rizvi A, et al. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *Lancet* 2013; **382:** 452–77.
2 Lelijveld N, Seal A, Wells JC, et al. Chronic disease outcomes after severe acute malnutrition in Malawian children (ChroSAM): a cohort study. *Lancet Glob Health* 2016; published online July 25. http://dx.doi.org/10.1016/S2214-109X(16)30133-4.
3 Kerac M, Bunn J, Seal A, et al. Probiotics and prebiotics for severe acute malnutrition (PRONUT study): a double-blind efficacy randomised controlled trial in Malawi. *Lancet* 2009; **374:** 136–44.
4 Christian P, Lee SE, Donahue Angel M, et al. Risk of childhood undernutrition related to small-for-gestational age and preterm birth in low- and middle-income countries. *Int J Epidemiol* 2013; **42:** 1340–55.
5 Jackson AA. Protein requirements for catch-up growth. *Proc Nutr Soc* 1990; **49:** 507–16.
6 FAO and WHO. Human energy requirements—report of a joint FAO/WHO/UNU expert consultation. 2001. http://www.fao.org/docrep/007/y5686e/y5686e00.htm (accessed June 16, 2016).
7 Semba RD, Shardell M, Sakr Ashour FA, et al. Child stunting is associated with low circulating essential amino acids. *EBioMedicine*. 2016; **6:** 246–52.
8 Golden MH, Golden BE. Effect of zinc supplementation on the dietary intake, rate of weight gain, and energy cost of tissue deposition in children recovering from severe malnutrition. *Am J Clin Nutr* 1981; **34:** 900–08.
9 Brenna JT, Akomo P, Bahwere P, et al. Balancing omega-6 and omega-3 fatty acids in ready-to-use therapeutic foods (RUTF). *BMC Med* 2015; **13:** 117.
10 Grantham-McGregor S, Powell C, Walker S, Chang S, Fletcher P. The long-term follow-up of severely malnourished children who participated in an intervention program. *Child Dev* 1994; **65** (2 Spec No)**:** 428–39.